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PART III

ENVIRONMENTAL PROTECTION AGENCY

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PESTICIDE PROGRAMS

Rebuttable Presumption Against Registration and Continued Registration of Pesticide Products Containing Cadmium

[6560-01]

ENVIRONMENTAL PROTECTION AGENCY

[FRL 807-6; OPP-30000/20]

PESTICIDE PROGRAMS

Rebuttable Presumption Against Registration and Continued Registration of Pesticide Products Containing Cadmium

The Deputy Assistant Administrator, Office of Pesticide Programs, Environmental Protection Agency (EPA), has determined that a rebuttable presumption exists against registration and continued registration of all pesticide products containing cadmium.[1]

I. Regulatory Provisions

A. *General.* Title 40, Part 162.11, of the Code of Federal Regulations for the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) as amended (86 Stat. 973, 89 Stat. 751, 7 U.S.C. 136 et seq.), provides that a rebuttable presumption against registration shall arise if the Agency determines that a pesticide meets or exceeds any of the risk criteria relating to acute and chronic toxic effects set forth in § 162.11(a)(3). If it is determined that such a rebuttable presumption has arisen, the regulations require that the registrant be notified by certified mail and afforded an opportunity to submit evidence in rebuttal of the presumption. In addition, the Agency has determined that the public should also be given notice of the bases for the presumption to provide an opportunity for comment and to solicit additional information relevant to the presumption.

A notice of rebuttable presumption against registration is issued when the evidence related to risk meets the criteria set forth in § 162.11(a)(3). It is emphasized that a notice of rebuttable presumption against registration and continued registration of a pesticide is not a notice of intent to cancel the registration of a pesticide, and may or may not lead to cancellation. The notice of intent to cancel is issued only if it is determined that the pesticide may generally cause unreasonable adverse effects to the environment after careful consideration of both the risks and benefits.

Accordingly, all registrants and applicants for registration are invited pursuant to 40 CFR 162.11(a)(4) to submit evidence in rebuttal of the presumptions listed in Part II of this notice and, in the case of oncogenicity, to submit information which relates to the assessment of oncogenic risks as set forth in the Agency's Interim Procedures and Guidelines for Health Risk and Economic Impact Assessment of Suspected Carcinogens (May 25, 1976; 41 FR 21402). Registrants and other interested parties may also submit for consideration data on benefits which they believe would justify registration or continued registration. In addition, any registrant may petition the Agency to voluntarily cancel a current registration pursuant to Section 6(a)(1) of FIFRA.

B. *Rebuttal Criteria.* Section 162.11(a)(4) provides that a registrant may rebut the presumption by sustaining the burden of proving:

(1) In the case of a pesticide presumed against pursuant to the chronic toxicity criteria, "that when considered with proposed restrictions on use and widespread and commonly recognized practices of use, the pesticide will not concentrate, persist or accrue to levels in man or the environment likely to result in any significant chronic adverse effects" (40 CFR 162.11(a)(4)(ii)); or

(2) That "the determination by the Agency that the pesticide meets or exceeds any of the criteria for risk was in error" (40 CFR 162.11(a)(4)(iii)).

C. *Benefits Information.* In addition to submitting evidence to rebut the presumption of risk, § 162.11(a)(5)(iii) provides that a registrant "may submit evidence as to whether the economic, social and environmental benefits of the use of the pesticide subject to the presumption outweigh the risk of use." If the risk presumptions are not rebutted, the benefit evidence submitted by the registrant,[2] applicants, and other interested persons will be considered by the Administrator in determining the appropriate regulatory action. Specifically, § 162.11(a)(5)(iii) provides that if the "benefits appear to outweigh (the) risks," the Administrator may issue a notice of intent to hold a hearing pursuant to Section 6(b)(2) of FIFRA rather than a notice of intent to cancel or deny registration pursuant to Section 3(c)(6) of FIFRA. Alternatively, if the "benefits do not appear to outweigh the risks, the Administrator shall issue a notice pursuant to Section 3(c)(6) or Section 6(b)(1) of the Act, as appropriate." Moreover, if at any time the Administrator determines that a pesticide poses an "imminent hazard" to humans or the environment, a notice of suspension may be issued pursuant to Section 6(c) of the Act.

Stated below are the § 162.11(a)(3) risk criteria which the Agency has found to have been met or exceeded by registrations and applications for registration of pesticide products containing cadmium. The Agency's basis for concluding that these risk criteria have been met or exceeded is set out in "Cadmium: Position Document 1," which follows. Copies of attachments to the Position Document which are not published with this notice are available for public inspection in the Office of Special Pesticide Reviews. Information protected from disclosure pursuant to Section 10 of FIFRA cannot be provided. Specific inquiries concerning the Position Document, as well as requests for access to these files, should be directed to the Project Manager, Mr. Richard Troast, Office of Special Pesticide Reviews (WH-566), EPA, Room 447, East Tower, 401 M Street SW., Washington, D.C. 20460, 202-755-8050.

II. Presumptions

A. *Oncogenicity.* 40 CFR 162.11(a)(3)(ii)(A) provides that a rebuttable presumption shall arise if a pesticide [i]nduces oncogenic effects in experimental mammalian species or in man as a result of oral, inhalation, or dermal exposure. * * *

On the basis of scientific studies summarized in the Position Document, the Agency has concluded that this risk index has been exceeded by all registrations and applications for registration of pesticide products containing cadmium, and that a rebuttable presumption against new or continued registration of such products has therefore arisen.

B. *Mutagenicity.* 40 CFR 162.11(a)(3)(ii)(A) provides that a rebuttable presumption shall arise if a pesticide "induces mutagenic effects, as determined by multitest evidence."

On the basis of scientific studies and information summarized in the Position Document, the Agency has concluded that this risk index has been exceeded by all registrations and applications for registration of pesticide products containing cadmium, and that a rebuttable presumption against new or continued registration of such products has therefore arisen.

C. *Other Chronic or Delayed Toxic Effects.* 40 CFR 162.11(a)(3)(ii)(B) provides that a rebuttable presumption shall arise if a pesticide "[p]roduces any other chronic or delayed toxic effect in test animals at any dosage up to a level, as determined by the Administrator, which is substantially higher than that to which humans can reasonably be anticipated to be exposed, taking into account ample margins of safety * * *."

On the basis of scientific studies and information summarized in the Position Document, the Agency has concluded that the risk index for teratogenicity and fetotoxicity has been exceeded by add registrations and applications for registration of pesticide products containing cadmium which are for home use, and that a rebuttable presumption against new or continued registration of such products has therefore arisen.

III. Additional Grounds for Review

As discussed in more detail in the attached Position Document, cadmium has been implicated as affecting a number of

[1] A position document containing an appendix of references, background information, and other material pertinent to the issuance of this notice, has been prepared by the Agency Working Group on cadmium and is also published with this notice.

[2] Registrants or other interested persons who desire to submit benefit information should consider submitting information on the following subjects, along with any other relevant information they desire to submit:

1. Identification of the uses of the pesticide, including estimated quantities used by application.
2. Identification of registered alternative products for the uses set forth in (1) above, including an estimate of their availability.
3. Determination of the change in costs to the user of providing equivalent pesticide treatment with any available substitute products.
4. Assessment of regulation impact upon user productivity from using available substitute pesticides or from using no other pesticides.

body systems, including lungs, kidneys, liver, cardiovascular, gonads, skeleton, hematopoietic, and central nervous systems. The data and analyses available at this time with respect to these effects are not sufficient to warrant the issuance of Rebuttable Presumption. The Agency specifically solicits further evidence bearing on these possible adverse effects. All comments and information received with respect to these potential adverse effects, including analysis thereof, may serve as a basis for a final decision as to the registerability of cadmium pesticides (40 CFR 162.11(a)(6)).

IV. Registration and Products Subject to the Notice

All registrants and applicants for registration listed below are being notified by certified mail of the rebuttable presumption existing against registration and continued registration of their products.

The registrants and applicants for registration shall have 45 days from the date this notice is sent or until December 12, 1977, to submit evidence in rebuttal of the presumption. However, the Administrator may, for good cause shown, grant an additional 60 days during which such evidence may be submitted. Notice of such an extension, if granted, will appear in the Federal Register.

A registrant or applicant for registration may, if it desires, assert a business confidentiality claim covering part or all of the information submitted in rebuttal. The registrant or applicant may assert the claim by placing on or attaching to the information a cover sheet, stamped or typed legend, or other suitable form of notice employing language such as "trade secret," "proprietary," or "company confidential." Allegedly confidential portions of otherwise nonconfidential documents should be clearly marked.

If a confidentiality claim is asserted, the information covered by the claim will be disclosed by EPA only to the extent and by means of the procedures set forth in 40 CFR Part 2, Subpart B (41 FR 36906, September 1, 1976). If no confidentiality claim accompanies the information at the time it is received by EPA, EPA will place the information in the public comment file where it will be available for public inspection.

If a registrant or applicant does assert a confidentiality claim for some, but not all, of the information submitted to EPA in rebuttal, the registrant or applicant should furnish two copies of the information to EPA. The first copy should contain all of the information submitted in rebuttal with information claimed as confidential clearly identified. The second copy should be identical to the first except that all information claimed as confidential should be deleted. The second copy will be placed in the public comment file. The first copy will be treated in accordance with the procedures set out above.

V. Duty to Submit Information on Adverse Effects

Registrants are required by law to submit to EPA any additional information regarding any adverse effects on man or the environment which comes to a registrant's attention at any time, pursuant to Section 6(a)(2) of FIFRA and 40 CFR 162.8(d). If any registrant of cadmium products has any published or unpublished information, studies, reports, analyses, or reanalyses regarding any adverse effects in animal species or humans, residues, and claimed or verified accidents to humans, domestic animals, or wildlife, which have not been previously submitted to EPA, the material must be submitted immediately. When responding to this notice, each registrant shall submit a written certification to the Agency that all information regarding any adverse effects known to the registrant has been submitted. In addition, the registrants should notify EPA of any studies currently in progress, including the purpose of the study, the protocol, the approximate completion date, and a summary of all results observed to date.

VI. Public Comments

During the time allowed for submission of rebuttal evidence, specific comments on the presumptions set forth in this notice and on the material contained in the Position Document are solicited from the public. In particular, any documented episodes of adverse effects to humans, domestic animals, or wildlife, and information as to any laboratory studies in progress or completed, are requested to be submitted to EPA as soon as possible. Specifically information on the degree of human exposure to cadmium and the possible effects thereof is solicited.

Similarly, any studies or comments on the benefits from the use of cadmium are requested to be submitted. All comments and information received, as well as any other relevant information and analysis thereof, which come to the attention of the Agency may serve as a basis for final determination pursuant to § 162.11(a)(5).

All comments and information should be sent to:

Federal Register Section, Technical Services Division (WH-569), Room 401, East Tower, 401 M Street SW., Washington, D.C. 20460.

Three copies of the comments or information should be submitted to facilitate the work of the Agency and others interested in inspecting them. The comments and information should bear the identifying notation "OPP-30000/20." Comments and information received within the specified time limit shall be considered before it is determined whether a notice shall be issued in accordance with 40 CFR 162.11(a)(5)(ii).

Comments received after the specified time period will be considered only to the extent feasible, consistent with the time limits imposed by 40 CFR 162.11(a)(5)(ii). All written comments and information filed pursuant to this notice will be available for public inspection in the office of the Federal Register Section from 8:30 a.m. to 4 p.m. during normal working days.

Interested persons are encouraged to take advantage of the opportunity to inspect Agency files during normal working hours since (1) all of the information received may serve as a basis for final determination pursuant to § 162.11(a)(5), and (2) the Agency will not generally publish a summary of information received in the Federal Register at the close of the rebuttal period.

Your cooperation is solicited in identifying any errors or omissions which may have been made in the following computer listings. Corrections to the listings may not necessarily be published in the Federal Register, but rather handled by mail with affected parties. Omissions will be corrected by notice in the Federal Register.

Dated: October 18, 1977.

Edwin L. Johnson,
Deputy Assistant Administrator for Pesticide Programs.

Cadmium: Position Document 1

Cadmium Working Group, Richard Troast, Project Manager, Office of Special Pesticide Reviews, U.S. Environmental Protection Agency

I. *Background*

A. *General Chemistry.* Cadmium occurs naturally, belongs to Group IIB in the periodic table of elements and has a molecular weight of 112.4. It constitutes 0.0001 percent of the earth's crust and is found primarily as the Mineral Greenockite (cadmium sulfide), which is not mined commercially but is extracted during zinc and lead mining operations. Electrolysis of smelted ore yields 99.9 percent pure metallic cadmium. The melting point and boiling point of elemental cadmium at 321° and 765°C, respectively; its density is 8.642 g/ml (1).

B. *Production.* About 12,000 pounds of pesticidal cadmium were produced in 1975. The production for 1976 is reported to have exceeded 20,000 pounds; that for 1977 is estimated to be as high as 40,000 pounds.

Section 7(c) of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) requires manufacturers and formulators to submit information on production, distribution, and sales of pesticidal cadmium compounds. Under Section 7(d) and 10 of FIFRA this information is considered confidential and may not be made available to the public.

The information is available to the Administrator in a confidential appendix to this report.

World-wide production of cadmium was reported as 17 million kilograms for 1975. To supplement domestic cadmium production, the United States imported 2.5 million kilograms in 1975 to meet an industrial demand of 5.6 million kilograms. Manufacture of cadmium pesticides accounted for less than 0.1 percent of the total amount of carmium used in the United States (2).

C. *Registered Uses.* Pesticidal cadmium compounds are registered as fungicides in sprays, dusts, and granular powders for application on ornamental turf, professional turf, and home lawns at a rate of 0.2 to 1.0 ounce of actual cadmium per 1000 square feet. Application rates vary depending on whether cadmium is applied alone or in combination with other fungicides.

There are 35 Federally registered cadmium products and one State registration for which Federal registration is pending (Table 1).

TABLE 1.—*Federal registrations and registrants of pesticidal cadmium compounds*

Compound	Registrations (number)	Registrants (number)
Cadmium chloride	*25	22
Cadmium carbonate	4	2
Cadmium sebecate	2	2
Cadmium succinate	3	3
Anilocadmium dilactate	1	1

*There is 1 pending Federal registration.

D. *Alternative Pesticides.* Alternative pesticides registered for use as fungicides on turf include dyrene, folpet, thiram, the ethylenebisdithiocarbamates, parachloronitrobenzene, captan, and benomyl. Of these, benomyl, the ethylenebisdithiocarbamates, and parachloronitrobenzene are themselves under review by the Office of Special Pesticide Reviews.

E. *Environmental Occurrence.*

1. *Soil.* Cadmium occurs in soil as a natural component, as a residue of fungicides applied directly to turf, and as a pollutant from industrial operations.

In soil samples taken from golf courses treated with cadmium fungicides, residues ranged from none detectable to 62.5 ppm. with a mean of 8.4 ppm. The greatest amounts were found in the top 4 inches of the soil, a fact that indiactes that cadmium does not migrate downward. Cadmium's translocation laterally through the soil is evidenced by its presence in the sediment of streams and ponds on the golf courses which have used cadmium pesticides. Half of the samples of sediment contained cadmium in amounts up to 94.4 ppm, with a mean of 1.5 ppm (3).

Industrial pollution from several sources deposits cadmium in the soil Cadmium particulates in the air settle to the ground and add measurably to the amount of cadmium in soils. The use of landfills for repositories of sludge creates areas of abnormally high concentrations of cadmium. The effluent from sewage treatment facilities contains 22 to 57 metric tons of cadmium per year (4). The concentration of cadmium averaged 3 ppm in the liquid effluent and 12 ppm in the sludge (5).

The amount of cadmium in soils is relatively low in nonindustrialized areas. Des Moines, Iowa, had means of 0.89 and 0.28 ppm in the urban and suburban areas, respectively, in contrast to Pittsburgh, Pa., an industrial center, which had a mean of 1.21 ppm for urban and 0.90 ppm for suburban areas (6).

2. *Air.* The amount of cadmium in the air depends on the degree of industrialization in a particular area. In 1969 survey, the concentration of cadmium in air ranged from 0.006 mg/m³ in San Francisco, Calif., to 0.036 mg/m³ in St. Louis, Mo. The highest concentration recorded in the United States, 0.12 mg/m³, was measured in 1964 in El Paso, Tex. (7). A 1970 survey, although limited, confirmed the 1969 study (7). IARC reported that contrations of cadmium worldwide average less than 0.003 mg/m³ in rural areas but as much as 0.050 mg/m³ in industrialized cities. In the vicinity of cadmium-emitting industries concentrations as great as 0.5 mg/m³ were recorded (2).

3. *Water.* Once deposited in water, cadmium is freely mobile (7). In rural areas, most cadmium comes from naturally occurring sources. In urban areas, naturally occurring cadmium is augmented by cadmium from industrial and commercial sources. Data from the Office of Water Supply in EPA indicate that cadmium is present in drinking water in amounts ranging from 0.01 ppm, the limit of detection, to 0.7 ppm.

4. *Food.* Cadmium (0.01 to 0.2 ppm) has been detected in foods of all categories monitored by FDA in its Market Basket Survey. The highest amounts were generally found in leafy vegetables and canned foods (8). The cadmium found in leafy vegetables probably does not result from application of cadmium pesticides because such pesticides are not registered for use on food crops.

The high concentrations found in green plants probably result from translocation of cadmium from soil, which is fortified by small amounts of cadmium impurities contained in phosphate fertilizers (9) and by unabsorbed cadmium ingested by animals and eliminated and redeposited in the soil or landfill (7).

5. *Other.* Humans are also exposed to cadmium in cigarette smoke. The IARC reports that each cigarette contains 1.7 μg of cadmium, up to 10 percent of which can be absorbed through the lungs into the circulatory system. In one year, a moderate smoker (one pack per day) may increase his total body burden of cadmium by 0.5 mg (2).

6. *Body Burden.* The total body burden varies with country and occupation. To arrive at a general mean, IARC (2) discounted occupational exposures and calculated the daily intake of cadmium from all other sources (table 2).

TABLE 2.—DAILY CADMIUM INTAKE

Country	Cadmium (μg/day)
Czechoslovakia	60
Japan	47–59
United States	4–92

Cadmium accumulates in sensitive target organs in which calcium has an important metabolic function. In the United States the average concentration of cadmium in the adult kidney was reported as 40 μg per gram wet weight, a value approximately one-half that for Japan (7).

Cadmium deposits in the body reach a maximum at about age 50 and thereafter decline logarithmically. The total body burden estimated at age 50 is 40 mg, the urinary excretion rate being less than 12 μg per liter of urine (10).

Once absorbed, cadmium is bound by metallothionein, a low molecular weight protein found in all organs associated with high concentrations of cadmium. Although metallothionein is thought to function in detoxifying or storing potentially toxic heavy metals, the amount of protection afforded is not known (7).

F. TOXICOLOGY

Cadmium has been the subject of a number of studies designed to identify and evaluate potential toxicological effects. These studies have used a variety of common cadmium compounds, some of which are not registered as pesticides. An EPA scientist at Research Triangle Park Laboratory advised the Cadmium Working Group that regardless of the cadmium salt used, the results observed can be attributed to the cadmium ion (11). Thus, the Cadmium Working Group has considered some studies which used cadmium compounds not registered as pesticides.

1. *Human.* Cadmium has been reported to affect many organ systems (7). A more detailed analysis of these effects in relation to exposure and dosage is addressed in Sections III and IV.

2. *Wildlife.* Data on the effects of cadmium to wildlife have not been reported in as much detail as the effects of cadmium on humans. A summary of some of the available data is presented below.

The LC_{50} for bluegills has been measured as 80μg/liter in water hardness of 200 mg/liter. Higher concentrations of cadmium did not inhibit spawning of surviving fish, but newly hatched progeny were deformed (12).

A study of the toxicity of cadmium to brook trout reported an LC_{100} of 6.4 μg of cadmium per liter in water hardness of 44 mg/liter. A concentration of 3.2 μg/liter killed the males either before or just after mating. The number of surviving embroys was smaller at this dosage. At a concentration of 1.7 μg/liter cadmium had no apparent effect on the reproductive capacity of the males or females (13).

The 96 hour LC_{50} of cadmium for flagfish was reported as 2500 μg/liter in water hardness of 4 mg/liter. The mortality of fish exposed to lower amounts of cadmium (31 μg/liter and 16 μg/liter) for up to 60 days was greater than that of controls. Growth of the fish exposed to cadmium in these concentrations was also reduced (14).

In 3-week-old chicks cadmium (9.5 mg/kg) interfered with the normal intestinal absorption of iron, causing an anemic condition typical of that found in mammals chronically exposed to low amounts of cadmium (15).

Japanese quail fed cadmium (75 mg/kg/day) for 6 weeks after hatching experienced various adverse effects when other minerals were deficient in their diet. Birds fed a diet deficient in zinc developed testicular hypoplasia, and their growth was retarded. Diets deficient in iron produced anemia and bone mar-

row hyperplasia. In both cases the heart was hypertrophied within 2 weeks after the experiment began (16).

The LC_{50} for quail fed formulated cadmium succinate was reported as 1700 mg/kg; that for mallard ducks was 5000 mg/kg (17).

Since cadmium pesticides are not applied directly to water, the hazard to aquatic species from normal application of cadmium pesticides seems minimal. The limited use of cadmium on home lawns and golf turf suggests that hazard to avian or mammalian species from cadmium pesticides would be similarly unlikely. Therefore, the Working Group has concluded that none of the data concerning toxicity to wildlife are sufficient to meet the criteria of 40 CFR 162.11(a)(3)(i)(B) and 162.11(a)(3)(ii)(c).

II. *Regulatory History*

Until 1968 cadmium compounds were registered both for use on turf and for limited use on food crops. Tolerances were established for these uses under the "no residue/zero tolerance" provisions of the regulations then in effect. After the U.S. Department of Agriculture discontinued the application of the "zero tolerance/no residue" principle, all registrations for the use of cadmium on food crops were cancelled by PR Notice 68–6, dated February 1, 1968, since data supporting finite tolerances were not submitted. The turf uses of cadmium were not affected by this notice.

The Administrator of EPA has placed a high priority on collection and review of data on cadmium (18). Many nonpesticidal uses of cadmium are subject to regulation under the Toxic Substances Control Act (TSCA) and regulatory action has been initiated under the Safe Drinking Water Act of 1974, which requires standards of purity in the Nation's drinking water supply. In its report to EPA the National Academy of Sciences recommended that the amount of cadmium in drinking water be limited to 10 μg/l (69). This standard was incorporated into the National Interim Primary Drinking Water Regulations which became effective on June 24, 1977 (70).

The National Cancer Institute is evaluating the oncogenicity of cadmium compounds injected into rodents.

The National Institute for Occupational Safety and Health (NIOSH) has recommended that occupational exposure not exceed 40 $\mu g/m^3$ per 10 hour day. NIOSH has also recommended that systems of medical examination, keeping medical records, sampling and analysis, area labeling, providing of protective clothing, training employees, and improving general work practices be used by industry (1).

III. *Summary of Scientific Evidence to Support Rebuttable Presumption: Chronic Toxicity*

40 CFR 162.11(a)(3) provides that a rebuttable presumption shall arise if a pesticide's ingredients, metabolites, or degradation products meet or exceed (i) acute toxicity risk criteria relating to hazards to humans, domestic animals, or wildlife, or (ii) chronic toxicity risk criteria relating to oncogenic, mutagenic, and delayed toxic effects in man or test animals, or relating to population reductions in nontarget organisms or fatalities to members of endangered species.

The Cadmium Working Group has concluded that pesticide products containing cadmium meet or exceed the chronic risk criteria relating to oncogenesis and mutagenesis. Certain uses of cadmium pesticides also meet or exceed the criteria relating to teratogenicity and fetotoxicity. Therefore, a presumption has arisen against all pesticides containing cadmium.

A. *Oncogenic Effects.* The Carcinogen Assessment Group (CAG) of EPA has reviewed laboratory studies on the oncogenic properties of cadmium administered orally and parenterally to animals and epidemiologic studies of human exposed to cadmium by inhalation or dermal contact. The CAG concluded that (i) the number of tumors at sites removed from the site of injection increased significantly in animals injected with cadmium; (ii) significant increases in prostate cancer were found in persons occupationally exposed to cadmium; and (iii) chronic oral studies, although reported as negative by their authors, must be considered inconclusive because of protocol deficiencies. The CAG also noted that some mutagenic actions of cadmium, including mammalian cellular transformations, have a high correlation with oncogenicity (19).

The CAG has considered all data bearing on the potential oncogenicity of cadmium regardless of the route of exposure. However, a rebuttable presumption on the basis of oncogenic effects arises only if such effects are observed in man or animals following oral, dermal, or inhalation exposure. The Working Group has concluded that the epidemiological data alone indicate that cadmium pesticides pose an oncogenic risk and are sufficient evidence to warrant a rebuttable presumption. The epidemiological studies are directly applicable to cadmium pesticide use since the routes of exposure in these studies (dermal and inhalation) are the same as the routes of exposure to cadmium pesticides.

Four occupational epidemiology studies (20, 21, 22, and 23) were reviewed by the CAG. In all of them, an association between prostate cancer and cadmium is suggested. In two of these studies, Kipling and Waterhouse (21) and Lemen et al. (22), the results obtained were statistically significant. The remaining two studies (20 and 23) suggest a correlation between cadmium exposure and excess prostate cancer mortality although no statistical analysis was completed. While these studies involved small numbers, the consistency of their findings raise a serious question as to the relationship of cadmium exposure and prostate cancer.

Berg and Burbank (24) reported that the presence of cadmium (3 μg/liter) in the drinking water supplies of the United States was positively correlated with the incidence of cancer in those areas. Cancers for which there was positive correlation included cancer of the pharynx, esophagus, intestine, larynx, lung, and bladder. The authors did not include the incidence of prostatic cancer among their data. A second drinking water study also indicated that a high concentration of trace metals, including cadmium, was positively correlated with the incidence of cancer in Czechoslovakia (25).

Accordingly a presumption has arisen against cadmium pesticides on the basis of the oncogenic effects observed in epidemiological studies.

B. *Mutagenic Effects.* The potential mutagenic effects of cadmium have been described in several comprehensive reviews and papers which have been evaluated by Agency scientists. Their analysis concludes that "cadmium does show the potential of interferring with the genetic material in living organisms" (26) and that "there is supportive evidence that cadmium may cause DNA damage leading to point mutations" (27). The valid studies indicate that cadmium is a point (gene) mutagen and can cause chromosomal damage.

Primary damage to DNA by cadmium has been observed in *in vitro* mammalian cell systems, bacteria and yeast. Chinese hamster embryo cells underwent unscheduled DNA synthesis after exposure to cadmium chloride and cadmium acetate, indicating an attempt by the cell to repair DNA damage presumably caused by the cadmium (28). Cadmium chloride induced a statistically significant increase of mispaired nucleotides when added to the *in vitro* DNA synthesis system (29). Cadmium chloride produced mutant recombinants using the recombinant assay test system in strains of *Bacillus subtilis*. Cadmium nitrate, which is not registered as a pesticide, was negative (30).

Cadmium chloride produced abnormal mitotic and meiotic recombinations in yeast, *Sacharomyces cerevisiae*, indicating that cadmium is capable of producing potential heritable damage in eukaryotic cells (31).

Cultured human leukocytes were exposed to cadmium sulfide for 4 to 8 hours. Cells examined microscopically for chromosomal aberrations revealed chromatid breaks, isochromatid breaks, translocations, and dicentrics (32). Similar chromosomal aberrations were found in human leukocytes from persons diagnosed with Itai-Itai disease. In addition the frequency of these aberrations was statistically greater in the Itai-Itai patients than in normal persons serving as controls (33).

Cadmium is capable of penetrating germinal tissue barriers and causing direct chromosomal damage. In an *in vivo* germinal cytogenetics test female mice were treated with an acute dose of cadmium chloride (3 or 6 mg/kg) and sacrificed after stimulation of ovulation. When examined microscopically, oocytes of treated animals revealed numerical chromosomal anomalies including hypoploidy, hyperploidy and diploidy. No such effects were observed in untreated

controls. Cadmium chloride also blocked development of metaphase I chromosomes in some of the animals and caused hemorrhaging of the ovaries (34).

Cadmium that penetrates the blood-testis barrier can affect development of mature sperm. When the rates of spermatogenesis in rats were measured at different times during development, more immature sperm cells were found after introduction of cadmium chloride than were found in controls. The rate of incorporation of radioactively labelled amino acid was also depressed, a fact that reflects a decreased rate of protein synthesis. The data also show a decreased fertility index for 50 days after the rats were injected intraperitoneally with cadmium at 1 mg/kg and then mated. After 50 days the fertility index returned to 80% of the initial levels (35). See also page 28 for additional gonadal effects.

The negative studies which were analyzed were either not sufficiently sensitive to mutagenicity for an accurate evaluation (36,37), lacked sufficient sample numbers (38), or were encumbered by other environmental factors which may have impacted on the stated results (39). A complete summary of all tests pertaining to mutagenicity is included in Table 3.

TABLE 3.—*Summary of tests pertaining to mutagenicity*

Test system	Genetic effect	Presence (+) or absence (−)	Appendix
Systems in vitro			
Chinese hamster cells	Point mutation	+	28
Polynucleotides	Base mispairing	+	29
Human cells	Chromosomal damage	+	32
B. subtilis—Recombinant assay	Gene mutation	+	30
S. cerevisiae	Point mutation	+	31
Systems in vivo			
Human	Chromosomal damage	+	33
Do	do	−	38
Rat	Altered spermatogenesis	+	35
Mouse	Dominant lethal mutations	−	36
Do	Cytogenetic damage	+	34
D. melanogaster	Sex-linked recessive lethal	−	37
Mammals	Chromosomal abnormalities	−	34

This evidence is ample to meet the definition of "multitest evidence" required by 40 CFR 162.11. The Working group has concluded, therefore, that a presumption has arisen against all pesticides containing cadmium on the basis of their mutagenic effects.

C. *Teratogenic Effects and Fetotoxic Effects.* Many publications have reported that cadmium is a potent animal teratogen and fetotoxic agent. For the purpose of this document 24 papers were reviewed by Battelle Memorial Laboratories, Columbus, Ohio. Their analysis concluded that cadmium is teratogenic in rats, mice, and hamsters (40). None of the studies which were reviewed established a no observable effect level.

Cadmium is transferred across the placenta to the fetus (41). This fact is extremely important because it demonstrates that cadmium can act directly upon the embryo, as well as indirectly by inducing toxic effects in the mother, which in turn might affect development. Battelle reviewed the available evidence and concluded that cadmium is a fetotoxic agent and that it inhibits postnatal survival and breeding.

Unlike most other chronic effects which manifest themselves very slowly over long periods, teratogenic and fetotoxic effects can be induced by one exposure occurring at a critical time. The evidence suggests that cadmium may exert such effects regardless of its route of administration or the time during gestation at which it is given.

When cadmium was administered to mice on day 7 of gestation, the adverse effects included decreased weight, exencephalia, excessive nucleic acid synthesis, and decreased growth of the brain (42). When cadmium was administered on days 14 to 17 of gestation, death of the embryo ensued attended by uterine hemorrhaging and other alterations in the vascular system (44). These data have been substantiated in a feeding study in which the death rates of the young and the production of runts of treated and control aniamls were significantly different. For lack of animals, the experiment was terminated with the F_2 generation (45).

In rats, the fetotoxic and teratogenic effects of cadmium (1.6 mg/kg in drinking water) included fetal mortality and decreased gross weight, hydrocephaly, cryptorchidism, hydronephrosis, and anophthalmia or microphthalmia (46). When rats were subcutaneously injected with cadmium (4 to 12 mg/kg), fetuses developed agnathia, cleft palate, and hypoplastic lungs (47). The differences in effects observed in these studies may be attributable to the different routes of administration and the days of treatment (gestation days 9 to 11 and 14 to 17, respectively).

In hamsters given cadmium intravenously (2 mg/kg given on gestation day 8) adverse effects included resorption of the fetus and formation of cleft palates and deformities in the primitive neural tube (exencephalia and microphthalmia) In another experiment, intravenous administration of cadmium at the same dose and time produced statistically significant gross malformations of the fore and hind limbs, including amelia, micromelia, and ectodactyly (48).

In a Germany study (41), women from three areas of differing degrees of industrialization were examined for cadmium levels in their placentas. Women in the most highly industrialized area, Rhine-Ruhr, had a mean level of 74.5 μg of cadmium in their placentas. This level was statistically higher than the two other lesser industrialized areas evaluated, Middle Frankonia (51.9 μg of cadmium) and Bavaria (55.1 μg of cadmium). This report did not evaluate the rate of miscarriage or the size of the children to assess any impact of cadmium on the pregnancy. However, another report stated that women occupationally exposed to cadmium did have abnormally small children (49). Because this study was not a controlled experiment, its data must be considered inconclusive.

Unlike the presumptions based on oncogenicity and mutagenicity, EPA regulations require that a presumption based on "other chronic effects" such as teratogenicity and fetotoxicity, must take into account human exposure in determining whether the criterion has been exceeded. The Working Group is unaware of any definitive data indicating the precise quantities of cadmium to which humans are exposed as the result of the use of cadmium pesticides. The Criteria and Evaluation Division (CED) of OPP has prepared a document titled "Preliminary Analysis of Human Exposure to Cadmium from the Use of Cadium Turf Fungicide" (50). In the absence of empirical evidence regarding human exposure, this analysis necessarily relies on certain assumptions, but its conclusions provide the best estimate available. The Working Group specifically solicits further exposure data, particularly from actual use.

In its analysis, the CED assumed that women of child-bearing age would only be exposed to cadmium pesticides from their use around the home. The professional use of cadmium pesticides on golf courses does not appear to result in significant exposure to women since only 6 of over 12,000 turf-maintenance supervisors are women (51). The CED document further assumes that as much as one pint of prepared cadmium spray could come in contact with the applicator during each use. Assuming 2% absorption to the sprays, the report concluded that an applicator could absorb as much as 0.8 mg of cadmium through dermal exposure to the sprays. Inhalation of a coarse water droplet was considered unlikely. In applying the dust a person could be exposed to as much as 0.8 mg of cadmium through combined dermal and inhalation exposure. The estimate assumes that normal clothing will limit dermal absorption to 0.5 mg and that the only inhalation exposure would result from the actual loading of the application equipment (0.3 mg of cadmium). Although cadmium pesticides are applied at most 12–14 times a year, only one exposure at a critical time is necessary to produce teratogenic and fetotoxic effects.

If a 60 kg woman were exposed to 0.8 mg of cadmium, she would receive a dose of 0.014 mg/kg. The Working Group does not consider the difference between this estimate of possible exposure and the lowest dosage which caused effects in the experimental studies great enough to ensure that no hazard will be posed to women that use cadmium pesticides. The difference becomes especially important since none of the studies reviewed demonstrated a no-effect level.

For the reasons stated above the Working Group concludes that the risk criterion is met and that a presumption based on teratogenicity and fetotoxicity has arisen against cadmium pesticides registered for home use.

IV. *Other Effects*

Cadmium is known to cause a number of adverse health effects in humans who have been exposed for relatively long periods of time to relatively high levels of cadmium. In addition, laboratory studies indicate that cadmium can cause a variety of adverse effects in test animals exposed to cadmium by several routes of exposure. However, it does not appear that any of the effects described below will occur at levels to which humans might reasonably be anticipated to be exposed from the use of pesticides, taking into account ample margins of safety. Moreover, the frequency and duration of exposure required to produce these effects does not seem comparable to exposure from application of cadmium pesticides. For these reasons the Cadmium Working Group has concluded that none of the effects discussed below warrants issuance of a presumption at this time.

A continuing effort will be made by the Working Group to collect additional data from all sources, including other regulatory groups and scientific organizations to determine if, with additional data, the criteria expressed in 40 CFR 162.11 are met for cadmium pesticides. In addition, the Agency, with the assistance of the United States Golf Association, has intiated a study of professional applicators of cadmium pesticides to determine if such persons exhibit signs of proteinuria, a condition often associated with excessive cadmium exposure and believed to be a possible indication of other adverse effects. If any of the data received or generated indicates potential risk, the Agency will consider additional regulatory action, including the issuance of a supplemental Notice of Rebuttable Presumption Against Registration.

The Working Group recommends solicitation of any additional information on the cadmium effects described in the following sections or any other effects which may be attributable to the pesticidal cadmium.

A. *Effects on the Lungs.* In humans, acute effects of doses of cadmium smaller than the estimated LD_{50} (approximately 2,400 mg-min/m^3) manifest themselves within 4 to 10 hours after exposure. Nonlethal effects of such doses in fumes or aerosols usually consist of pulmonary edema and interstitial pneumonitis from which damage in the form of perivascular and peribronchial fibrosis results (1, 7).

Emphysema has been observed in humans following lengthy exposure to low doses of cadmium (1, 7). Animal studies indicated that cellular changes typical of early stages of emphysema occur in rats given 17.2 μg/ml of cadmium in water daily for 6 to 41 weeks (52). Emphysema was induced in rats and guinea pigs by daily intratracheal administration of cadmium (2.5 mg/kg) for 4 and 7 months, respectively (53). A single intratracheal dose of cadmium chloride in an aerosol (9 mg/kg) induced preliminary symptoms of emphysema (54). However, an aerosol particle is much smaller and more readily absorbed than the coarse water droplet which would be encountered in the application of cadmium pesticides.

B. *Effects on the Kidneys.* Industrial workers accidentally inhaling high levels of cadmium fumes have suffered from acute renal necrosis (55). Temporary proteinuria has developed in humans exposed to subacute amounts cadmium for short periods (56), and renal failure is believed to have caused the death of a worker exposed to unmeasured levels of cadmium (20). Renal lesions, particularly formation of fatty bodies and degeneration of tubules, develop in animals exposed to large amounts of cadmium (1).

The effects of chronic exposure to cadmium are primarily proteinuria, glucosuria, excretion of amino acids in the urine, and decreased renal function (1). A specific protein, B–2 microglobulin, has recently been identified in the urine of Swedish workers and Japanese farmers exposed to cadmium (67).

When cadmium was given to rats in drinking water (0.2, 2.0, 20, and 200 ppm) changes (either constriction or dilation) to the renal arterial blood vessels resulted. These changes were not dose dependent, but seemed to be related to the dietary calcium levels. Rats with normal calcium levels in their diet experienced less alteration to the diameter of the blood vessels examined than those with low calcium levels (57).

Rats given cadmium in their drinking water (10, 50, and 300 ppm) for up to 40 weeks had an increased number of lysosomes in the kidney tubular epithelium. This increase was postulated by the authors to be related to the degeneration of renal tubules as previously theorized by other workers (43).

C. *Effects on the Liver and Carbohydrate Metabolism.* Early studies indicate that acute exposure to cadmium oxide fumes alters liver function. Autopsies have revealed microscopic changes in the liver, but these were slight compared to other acute effects (7).

The evidence suggests that workers chronically exposed to cadmium have impaired liver function (7). However, effects on the liver have not been examined as extensively as those on other organs.

Cadmium given intraperitonally to rats, either 1.0 mg/kg or 0.25 mg/kg every second day for 40 days, altered the glucose metabolic activity (58). Cadmium fed to rats, 1.0 mg/kg/day for 60 days, produced a statistically significant decrease in phosphorylase A enzyme activity. The same dosage resulted in a substantial decrease in the Pi/O ratio, suggesting an uncoupling of oxidative phosphorylation. In addition, a dose of 0.1 mg/kg/day for 42 days resulted in a significant decrease in aldolase activity (59). Insulin levels were significantly decreased in rats given seventy doses of 0.5 mg/kg cadmium every second day, whereas a 0.25 mg/kg dose did not produce these effects (58). In another study additional enzyme activities including ascorbic acid synthesis were altered in rats fed a dose of 60 mg/kg for four weeks (60).

D. *Effects on Cardiovascular System.* Cadmium in acutely toxic amounts increases the blood pressure in many rodent species (7), but there are no reports of this effect in exposed humans. However, epidemiologic evidence indicates that hypertension is less prevalent in areas of the world where the total body burden of cadmium is less (68).

Chronic human exposure has been epidemiologically linked to hypertension in some reports, but this is an area of some disagreement (7). Animal studies clearly indicate that hypertension is produced by cadmium given orally or by injection (1, 7). Rats given cadmium (5 ppm or 10 ppm for life) in their drinking water developed hypertension (62). Cadmium (5 ppm in drinking water) administered for the life of the animal induced arteriosclerosis by reducing the amount of circulating cholesterol (63).

E. *Effects on the Gonads.* When testicles were injected subcutaneously with cadmium (2.24 mg/kg), microscopic changes were evident within a few hours. Capillary stasis and interstitial edema were followed by severe hemorrhage and necrosis 24 to 48 hours later (7). Necrosis was dose-dependent and reversible.

Testicular necrosis also occurred when cadmium (28 and 64 mg/kg) was administered in food, painted on the skin, or injected intravenously or intraperitoneally (7). In a recent study, treatment with an amount of cadmium that produces general toxic effects (3 mg/kg in drinking water) also produced gonadotoxic effects including membrane thickening, hypertrophy and proliferation of Sertoli cells, and swelling of arteriolar walls (61). The lowest single subcutaneous dose which has produced acute testicular necrosis in the most sensitive laboratory animals, certain strains of mice, is 1 mg/kg (7).

Chronic effects on the gonads have been induced in animals fed cadmium or given repeated subcutaneous injections of cadmium. However, no testicular necrosis has been reported in humans industrially exposed to cadmium (1, 7).

F. *Effects on the Skeleton.* Cadmium is not known to accumulate in bone and correspondingly does not appear to have any acute effect upon the skeleton (67).

However, cadmium apparently affects the metabolism of calcium and phosphorus. Itai-Itai disease, probably the most insidious result of chronic exposure to cadmium in the food and water, is thought to have resulted from combined effects of interference of calcium and phosphous metabolism by cadmium, vitamin D deficiency, and a low protein diet. Low protein diets deficient in vitaman D are typical of Toyama Prefecture, Japan, the only area in which the disease has been found. Pseudo-fractures and decalcification have been found in humans exposed to cadmium for up to 20 years (1, 7).

Cadmium (5 and 10 ppm given in water for 50 days) causes tooth discoloration in rats (64). Bone disorders, in particular decalcification, develop in animals fed 1 to 2 mg/kg in food for 120 days (65).

G. *Effects on the Hematopoietic System.* Elevated hemoglobin concentrations are the only hematopoietic abnormality caused by acute exposure to cadmium. However, the edema that appeared concurrently may have been the primary result of exposure to cadmium, the elevated hemoglobin being only a consequence of the edema (7).

Chronic exposure to small quantities of cadium has been linked epidemiologically to decreased hemoglobin levels. White cell counts have been found to be essentially normal, with the exception of an increase in the number of eosinophiles. Bone marrow in all of these surveys showed no pathological changes. (7).

Animals treated with cadium have been shown to develop anemia and lowered hemoglobin levels. Dosages as low as 2.0 mg/kg fed to rats for four months have produced these effects. However, these effects can be mitigated by the simultaneous increase of additional iron to the diet. Dose response levels have been determined in rats given injections of cadium, but no dose-response levels have been determined in studies using other routes of exposure with levels ranging from 0.5 to 10 $\mu g/g$ for up to one year (7).

H. *Effects on the Central Nervous System.* Chronic cadium exposure produced headache and vertigo in one human (7). Physical examination revealed that he had altered neuromuscular reflexes. Olfactory disturbances, anosmia, and hyposmia were found to be common in a worker population, 50 percent of which had been exposed for at least 10 years at concentrations of 3 to 15 mg/m^3 (1).

Cadium injected intraperitoneally (0.25 and 1.0 mg/kg/day) for 45 days accumulated in the rat brain and acted on several indicators of brain activity such as the concentration of norepinephrine, acetylcholine, and 5-hydrotryptamine (66).

LIST OF APPENDICES

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08/23/77 FEDERALLY REGISTERED PRODUCTS CONTAINING CADMIUM

REGISTRANT *NAME AND ADDRESS*

* 000004 BONIDE CHEMICAL CO. INC.'
2 WURZ AVE.
YORKVILLE NY 13495

********** PRODUCT NAME **********

**00161* BONIDE LAWN FUNGICIDE

REGISTRANT *NAME AND ADDRESS*

* 000168 WASATCH CHEMICAL DIVISION ENTRADA IND INC
P O BOX 6219
SALT LAKE CITY UT 84106

********** PRODUCT NAME **********

**00331* MCGREO PLUS FUNGICIDE

REGISTRANT *NAME AND ADDRESS*

* 000239 CHEVRON CHEMICAL COMPANY
ORTHO DIVISION 940 HENSLEY WAY
RICHMOND CA 94801

********** PRODUCT NAME **********

**01426* ORTHO LAWN AND TURF FUNGICIDE

**01522* ORTHO LAWN DISEASE CONTROL

**02228* ORTHO GOLF AND TURF FUNGICIDE

08/23/77 FEDERALLY REGISTERED PRODUCTS CONTAINING CADMIUM

REGISTRANT *NAME AND ADDRESS*

* 000318 RUSSELL GERALD S
25-16 50TH ST
WOODSIDE LI NY 11377

********** PRODUCT NAME **********

**00039* RUSSELL'S GRANULAR LAWN FUNGICIDE

REGISTRANT *NAME AND ADDRESS*

* 000334 HYSAN CORPORATION
919 W 38TH ST
CHICAGO IL 60609

********** PRODUCT NAME **********

**00256* GCC-690 GROUND CONTROL CHEMICALS

REGISTRANT *NAME AND ADDRESS*

* 000358 NOTT MANUFACTURING COMPANY INC
PLEASANT VALLEY NY 12569

********** PRODUCT NAME **********

**00041* NOTT'S FUNGI-NCT, LAWN FUNGICIDE

REGISTRANT	*NAME AND ADDRESS*
* 000372	MALLINCKRODT INC SECOND AND MALLINCKRODT STREETS ST LOUIS, MO 63160

********** PRODUCT NAME **********

**00015* MALLINCKRODT CADMINATE

**00024* MALLINCKRODT IMPROVED KROMAD BROAD SPECTRUM FUNGICIDE

REGISTRANT	*NAME AND ADDRESS*
* 000603	CHEMTECH IND INC 3933 GLENFIELD, PO BOX 24440 DALLAS, TX 75224

********** PRODUCT NAME **********

**00027* GRASS-AID

REGISTRANT	*NAME AND ADDRESS*
* 000605	GUARD CHEM COMPANY ONE AVENUE L NEWARK NJ 07105

********** PRODUCT NAME **********

**00002* FURATURF 177

REGISTRANT	*NAME AND ADDRESS*
* 000702	ALPCO INCORPORATED P O 267 MARIETTA, OH 45750

********** PRODUCT NAME **********

**00024* CADEX A LIQUID CADMIUM TURF FUNGICIDE

REGISTRANT	*NAME AND ADDRESS*
* 001001	CLEARY W P CORPORATION 1049 SOMERSET ST. SOMERSET NJ 08873

********** PRODUCT NAME **********

**00010* CADDY

**00049* CAD-TRETE TURF FUNGICIDE

REGISTRANT	*NAME AND ADDRESS*
* 001159	SEACOAST LABORATORIES INC 257 HIGHWAY 18 E BRUNSWICK NJ 08816

********** PRODUCT NAME **********

**00136* TWIN LIGHT GRANULAR LAWN FUNGICIDE

**00182* TWIN LIGHT SNOW MOLD TURF FUNGICIDE

****

REGISTRANT *NAME AND ADDRESS*

* 001769 NATIONAL CHEMSEARCH DIV
USACHEM INC
2727 CHEMSEARCH BLVD
IRVING TX 75060

********** PRODUCT NAME **********

**00093* NATIONAL CHEMSEARCH C-A-D

****

REGISTRANT *NAME AND ADDRESS*

* 002124 GRACE W R & COMPANY AGR CHEM C H TIDWELL
PO BOX 277 100 N MAIN ST
MEMPHIS TN 38101

********** PRODUCT NAME **********

**00760* WONDER-GRO-TURF FUNGICIDE

****

REGISTRANT *NAME AND ADDRESS*

* 002474 HALCO DIVISION OF WAGNER SEED CO INC
81 SECATOGUE AVENUE
FARMINGDALE NY 11735

********** PRODUCT NAME **********

**00047* HALCO GRANULAR TURF FUNGICIDE

****

REGISTRANT *NAME AND ADDRESS*

* 002749 ACETO CHEMICAL COMPANY INC
126-02 NORTHERN BLVD
FLUSHING NY 11368

********** PRODUCT NAME **********

**00090* CADDY

**00127* ACETO CADET LIQUID CADMIUM FUNGICIDE

****

REGISTRANT *NAME AND ADDRESS*

* 002853 VINELAND CHEM COMPANY
PO BOX 745
VINELAND NJ 08360

********** PRODUCT NAME **********

**00010* VI-CAD A LIQUID CADMIUM TURF FUNGICIDE

****

REGISTRANT *NAME AND ADDRESS*

* 003442 USS AGRI-CHEMICALS DIV US STEEL CORP
PO BOX 1685
ATLANTA GA 30301

********** PRODUCT NAME **********

**00622* ARMOUR VERTAGARD BROAD-SPECTRUM K TURF FUNGICIDE

**00626* ARMOUR VERTAGARD CADMIUM TURF FUNGICIDE

REGISTRANT *NAME AND ADDRESS*

* 004828 ABCO INC
230 INDUSTRY BLVD
NORTH HUNTINGTON, PA 15642

********** PRODUCT NAME **********

**00080* TF-15 AN EFFICIENT TURF FUNGICIDE IN WETTABLE POWDER FORM

REGISTRANT *NAME AND ADDRESS*

* 005348 PATCO PRODUCTS INC
DIV OF VAUGHAN'S SEED CO
CHIMNEY ROCK ROAD
BOUND BROOK, NJ 08805

********** PRODUCT NAME **********

**00008* PATCO'S LAWN MEDIC A LAWN AND GRASS FUNGICIDE

REGISTRANT *NAME AND ADDRESS*

* 005535 ADIKES INC J & L
182-12 93RD AVE
JAMAICA NY 11423

********** PRODUCT NAME **********

**00002* GRO-WELL CRAB-N-WEED

**00034* GRO-WELL LAWN FUNGICIDE

REGISTRANT *NAME AND ADDRESS*

* 005605 FARMINGDALE GARDEN LABS
136 VERDI ST
FARMINGDALE NY 11735

********** PRODUCT NAME **********

**00085* FARMINGDALE CADMIUM LAWN FUNGICIDE (FORMULA B)

REGISTRANT *NAME AND ADDRESS*

* 006199 SANICO
13143 SATICOY ST
N HOLLYWOOD CA 91605

********** PRODUCT NAME **********

**00002* LF-34 LIQUID FUNGICIDE

REGISTRANT *NAME AND ADDRESS*

* 009235 AMERICAN RESEARCH CORPORATION
6899 PEACHTREE IND BLVD
NORCROSS, GA 30071

********** PRODUCT NAME **********

**00007* ARCIDE LIQUID CADMIUM CHLORIDE

REGISTRANT	*NAME AND ADDRESS*
* 010663	SENTRY CHEMICAL COMPANY 1481 ROCK MOUNTAIN BLVD STONE MOUNTAIN GA 30083

********** PRODUCT NAME **********

**00007* TF 33

REGISTRANT	*NAME AND ADDRESS*
* 011515	ABC CHEMICAL CORPORATION 17000 W. EIGHT MILE ROAD SOUTHFIELD, MI 48075

********** PRODUCT NAME **********

**00014* CUPRITE LIQUID TURF FUNG FOR CONTROL OF DOLLAR SPOT AND COPPER SPO

REGISTRANT	*NAME AND ADDRESS*
* 011694	DYMON, INC. 3401 KANSAS AVENUE KANSAS CITY, KS 66106

********** PRODUCT NAME **********

**00007* FCC 20 FUNGICIDE FOR CONTROLLING COPPER SPOT AND DOLLAR SPOT ON TURF

REGISTRANT	*NAME AND ADDRESS*
* 037347	UNI-CHEM CORP. OF FLORIDA BOX 6336 - 2801 N.W. 55TH CT. FT. LAUDERDALE, FL 33310

********** PRODUCT NAME **********

**03116* PREVENT

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